Electronic Version

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Claims

[c1] A method for inducing apoptosisin cancer cells, the method comprising the steps of contacting the living cells with a tyrosine kinase inhibitor and a histone deacetylase inhibitor.

[c2] The method of claim 1 wherein the tyrosine kinase inhibitor is imatinib mesylate.

[c3] The method of claim 1 wherein the histone deacetylase inhibitor is suberoylanilide hydromaxic acid.

[c4] The method of claim 1 wherein the tyrosine kinase inhibitor is imatinib mesylate and the histone deacetylase inhibitor is suberoylanilide hydromaxic acid.

[c5] Method of claim 1 wherein the living cells are exposed to the tyrosine kinase inhibitor and the histone deacetylase inhibitor for about 48 hours.

[c6] Method of claim 1 wherein the cancer cells are leukemia cells.

[c7] Method of claim 1 wherein the cancer cells are imatinib mesylate refractory.

[c8] A method of potentiating a cytotoxic effect of a tyrosine kinase inhibitor-based treatment comprising the steps of contacting target cells with a histone deacetylase.

[c9] The method of claim 8 wherein the tyrosine kinase inhibitor-based treatment comprises imatinib mesylate.

[c10] The method of claim 8 wherein the histone deacetylase inhibitor is suberoylanilide hydromaxic acid.

[c11] Method of claim 8 wherein the target cells are leukemia cells.

[c12] Method of claim 8 wherein the target cells are imatinib mesylate refractory.

[c13] A chemical composition for inducing apoptosis in cancer cells comprising a tyrosine kinase inhibitor and a histone deacetylase inhibitor.

[c14] The chemical composition of claim 13 wherein the tyrosine kinase inhibitor is imatinib mesylate.

[c15] The chemical composition of claim 13 wherein the histone deacetylase inhibitor is suberoylanilide hydromaxic acid.

[c16] The chemical composition of claim 13 wherein the tyrosine kinase inhibitor is imatinib mesylate and the histone

deacetylase inhibitor is suberoylanilide hydromaxic acid.